

Protein supplementation prevents etoposide-induced skeletal muscle damage

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Autophagy represents a physiological mechanism responsible for cell homeostasis and its deregulation is involved in several conditions related to muscle mass loss such as aging, inflammatory diseases and disuse [1]. In our previous work, double membrane vesicles, suggestive of autophagy, appeared after chemotherapeutic treatments in C2C12 myotubes [2]. Here, skeletal muscle cells have been exposed to Etoposide (Eto), a cell-death and oxidative stress inducer, as well as to protein supplementation before the trigger. Cytofluorimetric, morphological and molecular analyses revealed that Eto treatment increases cardiolipin peroxidation events, and induces lysosomal compartment and endoplasmic reticulum damage. Moreover, a peculiar accumulation of autophagic complex vacuoles resulted in LC3 localization into dot cytoplasmic structures, appeared in treated-differentiated cells, if compared to the diffuse cytoplasmic distribution observed in untreated cells. Protein supplementation, is able to prevent myotube damage, by reducing oxidative stress, improving the lysosomal degradation pathway, and, finally, by reactivating the protein synthesis. These findings suggest that a diet rich in protein could prevent the impaired autophagic degradation in a skeletal muscle model *in vitro*, exposed to a chemotherapeutic agent, thus contributing to delay the progression of several muscle disorders [3].

References

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Keywords

Skeletal muscle cells; etoposide; autophagy; protein supplementation.